U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 31 January 2006

In the Matter of:

GENEVA M. ALLEN,
Survivor of CHARLES F. ALLEN,
Claimant

v.

WESTMORELAND COAL CO.,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest

Case No.: 2003-BLA-6030

Appearances:

Joseph E. Wolfe, Esq.
Wolfe, Williams & Rutherford
Norton, Virginia
For the Claimant

Mary Rich Maloy, Esq.
Jackson Kelly, PLLC
Charleston, West Virginia
For the Employer/Carrier

Before: Alice M. Craft
Administrative Law Judge

DECISION AND ORDER DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 et seq. The Act and implementing regulations, 20 CFR Parts 410, 718, 725 and 727, provide compensation and other benefits to living coal miners who are totally disabled due to pneumoconiosis and their dependents, and surviving dependents of coal miners whose death was due to pneumoconiosis. The Act and regulations define pneumoconiosis, commonly known as black lung disease, as a chronic dust disease of the lungs and its sequelae, including

respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b); 20 CFR § 718.201 (2005). In this case, the Claimant, Geneva M. Allen, alleges that she is the surviving spouse of Charles F. Allen, whose death was due to pneumoconiosis.

I conducted a hearing on this claim on November 4, 2003, in Abingdon, Virginia. The parties were afforded a full opportunity to present evidence and argument, as provided in the Rules of Practice and Procedure before the Office of Administrative Law Judges, 29 CFR Part 18 (2005). The Claimant was unable to attend the hearing and no other witnesses provided testimony. Transcript ("Tr.") at 12-13. The Claimant's counsel requested that the case be submitted on the record and counsel for the Employer did not object. Tr. at 13. Director's Exhibits ("DX") 1-64 and Employer's Exhibits ("EX") 1-3, 5, 7 were admitted into evidence without objection. Transcript ("Tr.") at 6, 7, 12. Employer's exhibits 4 and 6 were excluded because they exceeded the limitations for the submission of evidence contained in the regulations, and the Employer failed to show good cause for their admission. Tr. at 12. The Claimant did not offer any exhibits. Tr. at 6. The parties made oral arguments, and the record was closed. Tr. at 19.

The evidentiary limitations applicable to this case were adopted in the new regulations effective January 19, 2001. Since the hearing, the Benefits Review Board has issued several significant decisions interpreting the effect of the evidentiary limitations contained in the new rules. Two in particular affect the record in this case. In *Smith v. Martin County Coal Corp.*, 23 B.L.R. 1-169 (2004), the Board held that the evidentiary limits are mandatory and cannot be waived by the parties. In *Church v. Kentland-Elkhorn Coal Corp.*, BRB Nos. 04-617 BLA and BLA-A (April 8, 2005), the Board held that medical evidence submitted in a living miner's claim is not automatically admissible in a survivor's claim filed after January 19, 2001. In this case, the miner's complete claim file was admitted as DX 1 without objection, and without discussion of the fact that it contained medical records which exceeded the limitations found in the new rules. In addition, duplicated records from DX 1 were included in DX 17, by the District Director, OWCP, and DX 28, by the Employer. The doctors consulted by the parties relied on records and reports from the miner's claim file, thus potentially violating the requirement of the rules that "Any chest X-ray interpretations, pulmonary function test results, blood gas studies, autopsy report, biopsy report, and physicians' opinions that appear in a medical report must each be admissible under this paragraph or paragraph (a)(4) of this section." 20 CFR § 725.414(a)(3)(i). Based on Dr. Perper's statement that he could not render an opinion as to the miner's cause of death without access to such records (*see* DX 30), however, I find that the parties had good cause for admission of the miner's claim file, and for their experts to refer to and rely on all of the available medical records. Thus I affirm admission into evidence of the miner's claim file, including all of the medical evidence, and have considered it in reaching my decision.

In reaching my decision, I have reviewed and considered the entire record, including all exhibits admitted into evidence and the arguments of the parties.

PROCEDURAL HISTORY

Mr. Allen filed a claim for black lung benefits on December 10, 1979. Although the District Director of the Office of Workers' Compensation Programs ("OWCP") made an initial finding of entitlement, his claim was eventually denied by the Benefits Review Board on February 28, 1994. Mr. Allen appealed to the U.S. Court of Appeals for the Fourth Circuit; however, the case was dismissed for want of prosecution on May 10, 1994. DX 1. Accordingly, Mr. Allen's claim for benefits is administratively closed and not subject to adjudication.

Mr. Allen died on September 26, 1999. DX 16. Mrs. Allen filed her claim for benefits on February 1, 2001. DX 2-4.[1] Her claim was initially granted by OWCP on February 27, 2003. DX 50. The Employer appealed, DX 52, 54 and 60, and the claim was transferred to the Office of Administrative Law Judges for hearing on May 28, 2003. DX 62.

ISSUE

The sole issue contested by the Employer before me is whether the miner's death was due to pneumoconiosis. DX 62; Tr. at 5.

APPLICABLE STANDARDS

A surviving spouse is entitled to benefits if the miner died due to pneumoconiosis which arose out of coal mine employment. *See* 30 U.S.C. § 901; 20 CFR §§ 718.205 and 725.212(a)(3) (2005). In this case, the Employer has stipulated that the miner suffered from pneumoconiosis arising from his coal mine employment. In claims filed after January 1, 1982, death will be considered to be due to pneumoconiosis if (1) competent medical evidence establishes that the miner's death was due to pneumoconiosis; (2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or (3) the presumption set forth at 20 CFR § 718.304 applies, i.e., an irrebuttable presumption that death was due to pneumoconiosis where there is medical evidence of complicated pneumoconiosis; but not if (4) the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 CFR § 718.205(c) (2005). The Fourth Circuit, in which this claim arises, has held that any condition that hastens the miner's death is a substantially contributing cause of death. *Shuff v. Cedar Coal Co*., 967 F.2d 977 (4th Cir. 1992). This principle has now been codified in the regulations at 20 CFR § 718.205(c)(5) (2005).

[1] Although the claim was originally filed in January 2001, that application was incomplete. The Claimant later submitted a complete application on February 1, 2001, and therefore her claim is considered to have been filed on that date. DX 2-4.

At hearing, the Employer conceded that the claim had been timely filed. The Employer also conceded that Claimant's spouse had been a miner with 33 years of coal mine employment[2] and that he had worked as a miner after December 31, 1969. The Employer conceded the presence of simple coal workers' pneumoconiosis ("CWP") arising out of coal mine employment and noted that disability was not at issue, since this is a survivor's claim. Finally, the Employer conceded that the miner had one dependent, his wife, Geneva, and that the responsible operator in this case was Westmoreland Coal Company, for whom the miner had last worked in 1980. Tr. at 5.

Medical Evidence

The records of both the miner's and survivor's claims contain treatment records and reports compiled in connection with the miner's claim from 1971 to 1996. DX 28, DX 17, DX 1. The record of the survivor's claim also contains treatment records from 1998 and 1999, DX 19-27, DX 29, as well as medical opinions on the cause of Mr. Allen's death. The physicians who gave opinions on the cause of death appear to have reviewed most or all of the medical evidence in both claims.

Treatment Records

Progress notes from the Big Stone Gap Clinic from May 1971 to January 1976 are found in DX 17 and DX 1. They are difficult to read, and do not appear to address pulmonary conditions for the most part. Mr. Allen had pneumonia in January 1975. Medical histories taken from Mr. Allen over the years indicate that he was told he had pneumoconiosis in 1973, but that diagnosis does not appear on his chart until January 1976.

Mr. Allen was examined by Dr. Joseph Smiddy at the request of his attorney in connection with his black lung claim on October 7, 1980. The results of that examination are reported below. In December 1984, Mr. Allen was admitted to the hospital for tests to rule out a right lung mass due to a change in his chest x-ray. CT scan did not show a specific well-defined mass, raising the possibility of atelectasis at the base of the right lung. Pulmonary function testing revealed a borderline obstructive defect, a mild restrictive defect, normal residual volume, and slightly decreased total lung capacity. Dr. Smiddy performed a bronchoscopy, resulting in a tentative diagnosis of chronic bronchitis. Discharge diagnoses were possible atelectasis involving a portion of the right lung, pneumoconiosis, chronic obstructive pulmonary disease ("COPD"), and arteriosclerotic heart disease ("ASHD"). On January 24, 1985, Dr. Smiddy wrote to Mr. Allen's attorney, stating:

> Based upon all of the information at hand and after detailed evaluation it is my opinion that this patient has sufficient Coal Workers' Pneumoconiosis to produce significant respiratory disability with significant arterial hypoxemia. This patient is not capable of performing the type of physical activity required for underground coal mine employment.

[2] The District Director found 37 years of coal mine employment. DX 62, 42, 32, and 13. The discrepancy has no significance for the outcome of the case. Thus I find that Mr. Allen had at least 33 years of coal mine employment.

DX 17, DX 1.

Mr. Allen's family doctor was Dr. Gary Williams, an internist. Dr. Williams' records for 1996 were in both claim files. At that time, Mr. Allen's visits to Dr. Williams were routinely scheduled four months apart. Dr. Williams' diagnoses included Type II diabetes and COPD alternating as the first and second diagnoses. In August 1996, Dr. Williams' notes indicate that Mr. Allen had been seen recently in the emergency room for possible pneumonia and possible congestive heart failure. After reviewing the results of an echocardiogram, Dr. Williams concluded Mr. Allen had had a heart attack in July. By November his congestive heart failure was well compensated. DX 17, DX 1. There is a three-year gap until the next records from Dr. Williams in the file.

Between January 1998 and June 1999, Mr. Allen saw Dr. Williams about every two months. Dr. Williams' diagnoses included ischemic cardiomyopathy, hypertension, compensated congestive heart failure, COPD, chronic renal failure, diabetes, and status post cerebrovascular accident ("CVA"), along with several others not relevant here. Dr. Williams reported an exacerbation of COPD in December 1998. In the first half of 1999, Dr. Williams' primary concerns related to Mr. Allen's significant weight loss and debilitation, and depression. On examination, his lungs were usually clear, although sometimes Dr. Williams reported diminished breath sounds, or a few wheezes or rhonchi. DX 27. The file also contains records of several hospitalizations in 1998 and 1999. Some of the records from those hospitalizations state that Mr. Allen was status post coronary artery bypass grafting, but the records of that surgery are not in the file; nor was it mentioned in Dr. Williams' progress notes.

Mr. Allen was hospitalized from August 20-22, 1998, due to congestive heart failure. DX 23.

Mr. Allen was evaluated for his weight loss and abdominal symptoms at Morton Community Hospital in April and May 1999. His workup was delayed due to a hydropneumothorax in April. DX 24.

Mr. Allen was admitted to the Norton Community Hospital from July 11 to 17, 1999, with pneumonia, placed on a ventilator, and then transferred to the Bristol Regional Medical Center for further evaluation. DX 21.

Mr. Allen was hospitalized at the Bristol Regional Medical Center under the care of Dr. John Byers from July 17 to August 9, 1999, due to respiratory failure and Methicillin resistant staph pneumonia. According to the discharge summary, he had "a long history of COPD related to tobacco abuse as well as history of coal workers pneumoconiosis." He was ventilator dependent when he was discharged, and went into a nursing home. The discharge plan was to remain on the ventilator as necessary, but wean as tolerated. DX 19-20.

Mr. Allen was hospitalized again from August 28, 1999, to September 10, 1999, due to heart arrhythmia and possible congestive heart failure. DX 20, DX 23, DX 26. Dr. J. Bryston Winegar was his attending physician. A consulting cardiologist, Dr. Keith Kramer, noted Mr. Allen's previous hospitalization for congestive heart failure in August 1998, when

echocardiogram showed his ejection fraction to be 45%. He was placed on medical therapy for ischemic cardiomyopathy at that time, which was felt to be the best choice for him due to his severe obstructive pulmonary disease. Dr. Kramer recommended continued medical management. Dr. Winegar's discharge summary described Mr. Allen as ventilator dependent, secondary to COPD, with chronic pneumonia. He was non-responsive, and unable to eat adequately, so a nasogastric tube was inserted. During his stay in the hospital, his arrhythmia improved, and he was discharged to the nursing home. The discharge diagnoses were right lower lobe pneumonia due to Methicillin resistant Staphylococcus aureus; tachybrady arrhythmia, improved; respiratory failure, ventilator dependent; multi-infarct dementia with dyspahgia; Type II diabetes mellitus; chronic obstructive pulmonary disease; chronic renal failure; prostatic hypertrophy; and stage 3 decubitus of sacrum. DX 26, DX 22-23. He died six days later.

Mr. Allen's death certificate is found at DX 16. He died on September 16, 1999, at age 81. The certificate was signed by Dr. Winegar, the attending physician during Mr. Allen's last hospitalization. Dr. Winegar listed the immediate cause of death as respiratory failure due to pneumonia, identifying two underlying causes that "initiated events resulting in death," namely, chronic obstructive pulmonary disease and coal workers' pneumoconiosis. He listed congestive heart failure and renal insufficiency as other significant conditions contributing to death.

A post mortem lung biopsy on the middle section of Mr. Allen's right lung was performed at Lonesome Pine Hospital on September 27, 1999 by Dr. Gary Adelson. His report states:

> On one surface of the specimen is a beige finely wrinkled membrane which may represent thickened visceral pleura or pericardium. The pleural surfaces are gray to dark purple-black, smooth and shiny. There are focal areas of subpleural emphysema … The lung parenchyma is light brown and diffusely mottled with gray-black areas. Some of these macules are located around the bronchioles. These macules … measure up to 5 mm in greatest dimension. In the subpleural region along one surface of the specimen are two gray-black nodules which measure up to 1.6 cm in greatest dimension … The bronchi contain gray mucus.

Dr. Adelson diagnosed "[a]nthracosilicotic macules and fibrocollagenous nodules compatible with simple (uncomplicated) coal workers' pneumoconiosis. Emphysematous change." DX 26, DX 18.

Additional Medical Evidence from Mr. Allen's Black Lung Claim

Chest X-rays

Chest x-rays may reveal opacities in the lungs caused by pneumoconiosis and other diseases. Larger and more numerous opacities result in greater lung impairment. The following table summarizes the x-ray findings available in connection with Mr. Allen's claim.

The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. Small opacities (1, 2, or 3) (in ascending order of profusion) may classified as round (p, q, r) or irregular (s, t, u), and may be evidence of "simple pneumoconiosis." Large opacities (greater than 1 cm) may be classified as A, B or C, in ascending order of size, and may be evidence of "complicated pneumoconiosis." A chest x-ray classified as category "0," including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 CFR § 718.102(b). Any such readings are therefore included in the "negative" column. In addition, several readers identified and classified opacities, but said they did not represent CWP; those readings are also included in the negative column. X-ray interpretations which made no reference to pneumoconiosis, positive or negative, given in connection with medical treatment or review of an x-ray film solely to determine its quality, have been omitted from this chart. Physicians' qualifications, where shown in the record, appear after their names. "B" indicates a NIOSH certified B reader; "BCR" indicates a board certified radiologist.

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis
01/22/75	DX 17, DX 1 Navani 1/1	
01/19/76	DX 17, DX 1 Navani 1/1	
04/11/80	DX 17, DX 1 Ramakrishnan 1/1 DX 17, DX 1 Fleenor 1/2	DX 17, DX 1 Wheeler B, BCR
10/07/80	DX 17, DX 1 Westerfield 2/2	
04/02/81	DX 17, DX 1 Evans 1	
12/05/84	DX 17, DX 1 Westerfield B, BCR 1/1	DX 17, DX 1 Scott B, BCR DX 17, DX 1 Wheeler B, BCR DX 28, DX 17, DX 1 Fino B DX 28, DX 17, DX 1 Morgan 0/1
09/30/85	DX 1, DX 17 DePonte B, BCR 1/1	DX 28, DX 17, DX 1 Scott B, BCR DX 17, DX 1 Wheeler B, BCR DX 28, DX 17, DX 1 Fino B DX 28, DX 17, DX 1 Morgan 0/1 DX 28, DX 17, DX 1 Wiot B, BCR (1/1, but not CWP) DX 28, DX 17, DX 1 Felson B, BCR (1/1, but not CWP) DX 28, DX 17, DX 1 Spitz B, BCR
12/06/85	DX 28, DX 17, DX 1 Spitz B, BCR 1/0 DX 17, DX 1 Robinette B 2/2	DX 17, DX 1 Scott B, BCR DX 17, DX 1 Wheeler B, BCR DX 28, DX 17, DX 1 Fino B DX 28, DX 17, DX 1 Morgan 0/1 DX 17, DX 1 Wiot B, BCR
08/07/86		DX 17, DX 1 Scott B, BCR DX 17, DX 1 Wheeler B, BCR DX 28, DX 17, DX 1 Fino B

Date of X-ray	Read as Positive for Pneumoconiosis	Read as Negative for Pneumoconiosis
09/22/86	DX 17, DX 1 Westerfield B, BCR 1/1	DX 28, DX 17, DX 1 Felson B, BCR DX 28, DX 17, DX 1 Spitz B, BCR DX 17, DX 1 Scott B, BCR DX 17, DX 1 Wheeler B, BCR DX 28, DX 17, DX 1 Fino B
08/14/90	DX 17, DX 1 Mathur B, BCR 1/2 DX 17, DX 1 Westerfield B, BCR 1/1	DX 28, DX 17, DX 1 Scott B, BCR DX 17, DX 1 Wheeler B, BCR DX 28, DX 17, DX 1 Fino B DX 17, DX 1 Hippensteel B DX 28, DX 17, DX 1 Wiot B, BCR

Pulmonary Function Studies

Pulmonary function studies are tests performed to measure obstruction in the airways of the lungs and the degree of impairment of pulmonary function. The greater the resistance to the flow of air, the more severe the lung impairment. The studies range from simple tests of ventilation to very sophisticated examinations requiring complicated equipment. The most frequently performed tests measure forced vital capacity (FVC), forced expiratory volume in one-second (FEV_1) and maximum voluntary ventilation (MVV).

The following chart summarizes the results of the pulmonary function studies available in connection with the miner's claim. "Pre" and "post" refer to administration of bronchodilators. If only one figure appears, bronchodilators were not administered. In a "qualifying" pulmonary study, the FEV_1 must be equal to or less than the applicable values set forth in the tables in Appendix B of Part 718, and either the FVC or MVV must be equal to or less than the applicable table value, or the FEV_1/FVC ratio must be 55% or less. 20 CFR § 718.204(b)(2)(i).

Ex. No. Date Physician	Age Height[3]	FEV_1 Pre-/ Post	FVC Pre-/ Post	FEV_1/ FVC Pre-/ Post	MVV Pre-/ Post	Qualify?	Physician Impression
DX 17, DX 1 04/07/80 Fleenor	61 70"	2.98	4.27	70%	150	No	Within normal limits

[3] The fact-finder must resolve conflicting heights of the miner recorded on the ventilatory study reports in the claim. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221, 1-223 (1983); *Toler v. Eastern Assoc. Coal Co.*, 43 F.3d 109, 114, 116 (4th Cir. 1995). As there is a variance in the recorded height of the miner from 68.25" to 70", I have taken the average (69.2") in determining whether the studies qualify to show disability under the regulations. None of the tests are qualifying to show disability whether considering the average height, or the heights listed by the persons who administered the testing.

Ex. No. Date Physician	Age Height[3]	FEV_1 Pre-/ Post	FVC Pre-/ Post	FEV_1/ FVC Pre-/ Post	MVV Pre-/ Post	Qualify?	Physician Impression
DX 17, DX 1 06/13/80 Kanwal	62 69”	2.35	3.15		79.8	No	Compatible with obstructive and restrictive disease. Invalid per Drs. O’Neill, Zaldivar
DX 17, DX 1 04/02/81 Abernathy	62 70”	2.88 3.18	3.79 4.25	76% 75%	94 90	No No	Reduced MVV. Otherwise normal.
DX 17, DX 1 12/11/84 Smiddy	66 69”	2.97 2.49	3.20 3.25	73% 77%	120 123	No No	Borderline obstructive defect, mild restrictive defect.
DX 17, DX 1 12/06/85 Robinette	67 69”	2.40	3.54	67%	92	No	Normal. Invalid per Drs. Vest, Renn.
DX 17, DX 1 08/07/86 Dahhan	68 68.25”	2.4 2.3	3.35 3.35		106.9 103.9	No No	Normal

Arterial Blood Gas Studies

Blood gas studies are performed to measure the ability of the lungs to oxygenate blood. A defect will manifest itself primarily as a fall in arterial oxygen tension either at rest or during exercise. The blood sample is analyzed for the percentage of oxygen (PO_2) and the percentage of carbon dioxide (PCO_2) in the blood. A lower level of oxygen (O_2) compared to carbon dioxide (CO_2) in the blood indicates a deficiency in the transfer of gases through the alveoli which may leave the miner disabled.

The following chart summarizes the arterial blood gas studies available in connection with the miner’s claim. A “qualifying” arterial gas study yields values which are equal to or less than the applicable values set forth in the tables in Appendix C of Part 718. If the results of a blood gas test at rest do not satisfy Appendix C, then an exercise blood gas test can be offered. Tests with only one figure represent studies at rest only. Exercise studies are not required if medically contraindicated. 20 CFR § 718.105(b).

Exhibit Number	Date	Physician	PCO_2 at rest/ exercise	PO_2 at rest/ exercise	Qualify?	Physician Impression
DX 17, DX 1	04/21/80	Paranthaman	39.7 33.6	95.1 74.8	No No	Moderate resting hypoxemia improving on exercise.
DX 17, DX 1	06/13/80? 09/08/80	Kanwal	35.8 35.8	63.4 79.0	Yes No	Hypoxemia.
DX 17, DX 1	12/11/84	Smiddy	38.8	67.6	No	
DX 17, DX 1	12/06/85	Robinette	33.3	60.5	Yes	Moderate resting hypoxemia
DX 17, DX 1	08/07/86	Dahhan	36.9 38.1	73.5 78.7	No No	Adequate ventilation

Medical Opinions

Medical opinions are relevant to the issues of whether the miner had pneumoconiosis, whether the miner was totally disabled, and whether pneumoconiosis caused the miner's disability and death. The medical opinions must be reasoned and supported by objective medical evidence such as blood gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. 20 CFR § 718.202(a)(4). The record contains the following medical opinions relating to the miner's claim.

Ex. No./ Date of Examination or Review of Records	Physician/ Basis for Opinion	Opinion Regarding Existence of Pneumoconiosis	Opinion Regarding Lung Impairment or Disability and Cause
DX 17, DX 1 04/11/80	Fleenor Examined	Has black lung.	
DX 17, DX 1 06/13/80	Kanwal Examined	Has CWP	Pulmonary condition prevents miner from engaging in coal mine or comparable work.
DX 28, DX 17, DX 1 04/03/81 04/27/81	Abernathy Examined and reviewed records	Has CWP	Major problem from chronic bronchitis due to cigarette smoking. No impairment of oxygen transfer. Not disabled.
DX 17, DX 1 10/07/80 01/24/85	Smiddy Examined	Has CWP	CWP produces significant respiratory disability. Disabled.

Ex. No./ Date of Examination or Review of Records	Physician/ Basis for Opinion	Opinion Regarding Existence of Pneumoconiosis	Opinion Regarding Lung Impairment or Disability and Cause
DX 28, DX 17, DX 1 07/07/81 08/05/83 07/01/87 08/17/87 09/09/90	Kress Reviewed records	Could not rule out very early CWP	No impairment due to CWP. Impairment secondary to chronic bronchitis due to smoking, but not significant or persistent. Not disabled by it, although he may be due to other unrelated factors.
DX 17, DX 1 Undated 12/31/85 10/23/90	Robinette Examined and reviewed records	Has CWP	Disabled due to varying levels of hypoxemia and persistent respiratory problems caused by CWP and emphysematous change.
DX 18, DX 17, DX 1 08/07/86 08/28/90 12/10/90	Dahhan Examined and reviewed records	No evidence of CWP	Mild obstructive impairment caused by smoking, not disabling. No evidence of total or permanent pulmonary disability secondary to coal dust exposure
DX 28, DX 17, DX 1 09/19/90 12/13/90	Fino Reviewed records	Does not suffer an occupationally acquired pulmonary disorder	No respiratory impairment. Not disabled.
DX 28, DX 17, DX 1 05/26/87 08/24/87 08/28/90 12/14/90	Morgan Reviewed records	No.	Normal ventilatory capacity. Not disabled. May have minimal ventilation perfusion mismatching from obesity.
DX 28, DX 17, DX 1 05/27/87 08/19/87 09/08/90 12/15/90	Tuteur Reviewed records	May have CWP, but it is not associated with clinical symptoms or impairment of pulmonary function	Has a respiratory impairment but it is not caused by CWP and he is not disabled based on it alone.
DX 28, DX 17, DX 1 09/18/90 12/17/90	Stewart Reviewed records	Does not have CWP	Does have a mild obstructive impairment, but not disabling

Opinions on the Cause of Mr. Allen's Death

Dr. Perper's Opinion

Dr. Joshua A. Perper, a forensic pathologist and medicolegal consultant, prepared a report dated December 26, 2001, at the request of the Department of Labor. DX 30. Dr. Perper had previously submitted a report in August 2001, addressing just the records from 1998 and 1999, but said he had insufficient information to form an opinion. He was then provided with records for the period from 1971 to 1990 from the miner's claim. Apparently Dr. Perper's earlier report was removed from the file as it was thought to be excessive under the evidentiary limitations. *See* DX 30, 41.

In his December report, Dr. Perper listed the additional information he had reviewed, and noted Mr. Allen's occupational history of at least 30 years in the mines ending in 1980, and a smoking history of 40 pack years ceasing in 1988, as reported in the medical records. He summarized Mr. Allen's medical history as reflected in clinic notes and black lung examinations from 1971 to 1990. Dr. Perper did not reiterate his summary of the 1998-1999 records apparently contained in the missing August report, but said that the historical documentation provided the additional information he needed to form an opinion.

Dr. Perper concluded that Mr. Allen had pneumoconiosis based on

> -Clinical symptomatology for many years of shortness of breath, cough, expectoration of mucus, with objective findings on clinical examination of barrel shaped chest, abnormal respiratory breathing sounds, and impaired pulmonary functions and hypoxemia. The respiratory symptomatology developed many years before any evidence of cardiac failure was evident, and became most pronounced in the miner's [last] two years of life with treatment for pulmonary disability to respiratory failure with need for bronchodilators and eventually a need for supplemental oxygen, and respirator dependency.
> As a matter of fact a number of clinical diagnoses mentioned coal workers' pneumoconiosis as the cause of the respiratory symptomatology.
>
> -Radiological evidence for many years, starting in the 1970s of pulmonary opacities, which were interpreted by many qualified readers as diagnostic for simple coal workers pneumoconiosis.
>
> The examiners who denied the presence of … pneumoconiosis, speculated that the pulmonary nodules/opacities were granulomas [caused by other disease processes for which there was no clinical or laboratory evidence]
> The autopsy specimen showed only clear evidence of coal workers' pneumoconiosis with no other pathological findings of other granulomas … and therefore it seems quite evident that the so called granulomas were in fact lesions of coal workers' pneumoconiosis …
>
> -The post mortem autopsy specimen of Mr. Allen, revealed unquestionable presence of significant simple coal workers' pneumoconiosis both gross and microscopic, as

> documented by Dr. … Adelson … The microscopic examination by the current reviewer, verified the presence of simple, slight to moderately severe coal workers' pneumoconiosis mostly macular and micronodular, but with an occasional silicotic macronodule, scar emphysema and presence of birefringent silica crystals.
> It is well known that the pathologic examination is the *golden standard* for the diagnosis of coal workers' pneumoconiosis.
> …

DX 30:21-22. Dr. Perper went on to state that Mr. Allen's pneumoconiosis resulted from occupational exposure to coal dust.

Dr. Perper next addressed the question of whether, generally, exposure to coal mine dust and coal workers' pneumoconiosis can result in centrilobular emphysema. He stated, "The causal connection between exposure to coal and silica in regard to emphysema and chronic obstructive lung disease is widely and universally accepted." DX 30:23. He went on to cite medical literature in support of his premise, including Gregory Wagner, the Director of the Division of Respiratory Disease of the National Institute of Occupational Safety and Health, *Screening and Surveillance of Workers Exposed to Mineral Dust*, 1996, WHO-Geneva, who said, "'Chronic bronchitis, airflow limitations, CWP and *emphysema* (italics by G. Wagner) all result from exposure to coal mine dust and may occur in various combination.'" In addition, Dr. Perper cited to a publication by the U.S. Department of Health and Human Services stating, "'COPD refers to three disease processes – chronic bronchitis, emphysema and asthma – that are all characterized by airway dysfunction … A major cause of COPD is cigarette smoking; but air pollution and occupational exposure to dust, particularly among smokers, can also cause COPD.'" DX 30:23 *citing* U.S. Department of Health and Human Services, the Public Health Service, the Centers for Disease Control and Prevention, and NIOSH, *Criteria for a Recommended Standard – Occupational Exposure to Coal Mine Dust*, September 1995.

Citing the same September 1995 publication, Dr. Perper referenced a section entitled "Studies of COPD in Coal Miners, Four American Studies Published Between 1982 and 1992" showing that exposure to respirable dust is associated with decrements in lung function among coal miners. He then cited a section entitled "Emphysema" that includes five autopsy studies on U.K. coal miners and two Australian studies between 1982 and 1994 showing "'a significant increase in emphysema among coal miners as compared with non-mining populations.'" Dr. Perper noted that some of the studies quoted controlled for smoking, and concluded that "'the relationship with dust exposures was only apparent among those with centriacinar emphysema'" and that "'the extent of emphysema in smokers was significantly related to both coal dust of the lungs and to smoking.'" By contrast, in "'non-smokers, the extent of emphysema was significantly related to both the coal dust content of the lung and age.'"

Dr. Perper asserted that, in fact, more recent studies have concentrated "not on the already proven relationship between exposure to coal mine dust and centriacinar (centrilobular) emphysema but on elucidating the mechanism of coal dust toxicity and how it compares to smoking in causing the emphysema." DX 30:23. Specifically, he cited a 1997 report by Canadian researchers, which states that "[m]ineral dust exposure can result in emphysema and chronic airflow obstruction"; Dr. Perper noted that this study "included both in vitro and in vivo

animal experimentation of toxicity of quartz and coal, which supports the assumption that dust induced emphysema and smoke induced emphysema occur through similar mechanisms." DX 30:23-24 *citing* Churg A., Katalin Z., and Kevin L., *Mechanisms of Mineral Dust-Induced Emphysema*, Environ. Health Perspect. 105, Supplement 5, September 1997. Dr. Perper also mentioned that the Act's implementing regulations "clearly support the causal connection between the exposure to coal mine dust and coal workers' pneumoconiosis and the development of chronic obstructive lung disease and centrilobular emphysema, relying on an extensive review of the literature by NIOSH." DX 30:24.

Dr. Perper also cited literature stating that "[i]n several investigations, emphysema has been shown to be present more often and to be more advanced in patients with CWP than in those without it; the severity correlates with the degree of exposure to coal dust and has been demonstrated to be independent of age and cigarette smoking and to be positively related to the dust content of the macules." DX 30:24 *citing* R.S. Fraser, J.A. Peter Pare, R.G. Fraser, P.D. Pare, *Synopsis of Diseases of the Chest*, Second Edition, W.B. Saunders Company, 1998. He further noted that a recent article on occupational respiratory diseases indicates that "common causes of occupational airway disease, included chronic obstructive pulmonary disease and chronic air flow limitation due to 'coal dust (causes emphysema with nodular fibrosis) and crystalline silica (causes chronic airflow limitation).'" DX 30:24 *citing* William S. Beckett, *Occupational Respiratory Disease*, New Engl. J. Med. 2000, Feb; 342(6):406-413. Dr. Perper also noted that in England and Germany, chronic bronchitis and/or emphysema of coal miners have been included in the list of occupational diseases. DX 30:24.

After citing the medical literature confirming his belief that coal mine dust and coal workers' pneumoconiosis can result in centrilobular emphysema, Dr. Perper then applied the medical literature to the case of Mr. Allen. He noted that Mr. Allen had had a clinical history of significant cigarette smoking for many years but quit in 1988. Despite his having quit 11 years before his death, Dr. Perper admitted that cigarette smoking was "a substantial factor in the causation of Mr. Allen's centrilobular emphysema." However, Dr. Perper further posited: "[O]ne cannot exclude the equally significant impact of occupational exposure to coal mine dust and coal workers' pneumoconiosis." DX 30:24.

Next, Dr. Perper addressed the question of whether coal workers' pneumoconiosis progresses in severity after cessation of occupational exposure to coal mine dust. DX 30:25. He opined that this is the case, stating that "recent literature has substantiated that the pulmonary damage associated with exposure to coal mine dust and coal workers' pneumoconiosis may progress even after the cessation of occupational exposure to silica containing mixed coal mine dust. Dr. Perper further stated: "This is easily appreciated if one consider [sic] that the inhaled silica remains trapped in the lungs and cannot be removed." To substantiate this idea, Dr. Perper cited the statistic that among 1902 ex-miners who had not developed complicated coal workers' pneumoconiosis within 4 years of leaving mining, 172 (9%) developed it after mining. Of those 172 miners with complicated coal workers' pneumoconiosis, 32% had no evidence of simple CWP (category 0) when they left mining. DX 30:25 *citing* Maclaren WM, Soutar CA, *Progressive Massive Fibrosis and Simple Pneumoconiosis in Ex-Miners*, Br. J. Med. 42:734-740, 1985. Dr. Perper cited various sources to further substantiate the progressivity of pneumoconiosis, including the final rule published by the U.S. Department of Labor on

December 20, 2000, accepting that coal workers' pneumoconiosis may progress after cessation of exposure. With respect to Mr. Allen's case specifically, Dr. Perper stated that his clinical history "clearly indicates a worsening of his respiratory symptomatology and findings after he ceased smoking." DX 30:25.

Finally, Dr. Perper addressed "whether coal workers' pneumoconiosis was a substantial contributory cause of Mr. Charles Allen's death or a hastening factor in his death, and if yes, by what mechanism." Dr. Perper stated: "Coal workers' pneumoconiosis was a substantial contributory cause of Mr. Allen's death both directly and indirectly through the associated centrilobular emphysema that caused hypoxemia resulting ultimately in respiratory failure and requiring the administration of bronchodilators and oxygen." He also stated: "An additional mechanism of death of coal workers' pneumoconiosis and associated COPD, was the causation or precipitation of a fatal arrhythmia in a patient with coronary arteriosclerosis and atrial fibrillation." He said Mr. Allen's "[c]oal workers' pneumoconiosis demonstrated at autopsy with the associated centrilobular emphysema was of sufficient extent and severity to constitute a substantial contributory cause of Mr. Allen's death, both directly through the marked pneumoconiotic process and indirectly through associated centrilobular emphysema, that caused hypoxemia that either triggered or aggravated a fatal cardiac arrhythmia in an individual with marked arteriosclerotic heart disease." DX 30:26. In conclusion, he noted that the "scientific literature supports such mechanism" citing that "'patients with chronic obstructive pulmonary disease (COPD), especially during acute exacerbations of their disease show a greater incidence of cardiac arrhythmia than healthy subjects of the same age.'" DX 30:26 *citing* Conte G, Lauro S, Lazzarin M, Rigon N, Perrone A, *Evaluation of Hyperkinetic Cardiac Arrhythmia in Chronic Obstructive Bronchopneumopathy*, Minerva Cardioangiol 1997 Sep: 45(9)429-33. Similarly, other literature notes that "'patients with chronic obstructive lung disease have a high incidence and wide variety of cardiac arrhythmias.'" DX 30:26 *citing* Biggs FD, Lefrak SS, Kleiger RE, Senior RM, Oliver GC, *Disturbances of Rhythm in Chronic Lung Disease*, Heart Lung 1997 Mar-Apr; 6(2):256-61.

Dr. Perper's conclusions were as follows: (1) Mr. Allen had evidence of mild to moderately severe coal workers' pneumoconiosis with associated centrilobular emphysema; (2) Mr. Allen developed coal workers' pneumoconiosis as a result of his long standing occupational exposure to mixed coal mine dust containing silica; and (3) Coal workers' pneumoconiosis with related centrilobular emphysema was a substantial contributory cause of Mr. Allen's death, both directly and through hypoxemia, and through causing, precipitating or aggravating a fatal arrhythmia in a patient with arteriosclerotic heart disease. DX 30:27.

Dr. Caffrey's Opinion

Dr. P. Raphael Caffrey generated a Pathology Consultation Report dated February 7, 2003, EX 2, offered by the Employer to rebut Dr. Perper's opinion. Dr. Caffrey is a board-certified pathologist. EX 2. He noted that Mr. Allen had approximately 37 years of underground coal mine employment, and last worked on June 27, 1980. He calculated a 50-year history of smoking one pack of cigarettes daily. EX 2:3.

In preparing his report, Dr. Caffrey reviewed several aspects of Mr. Allen's medical history, including treatment records and medical reports of other physicians. *See* EX 2:1-2. Among other things, Dr. Caffrey considered Dr. Perper's December 26, 2001 report, Mr. Allen's death certificate, Dr. Gary L. Adelson's pathology report dated October 1, 1999, and ten surgical pathology slides.[4] *See* EX 2:2.

Dr. Caffrey noted that there is no clinical information on the one-page surgical pathology (autopsy) report. It appears that the right lung biopsy was taken by Michael B. Ford, MD, and sent to Dr. Adelson, Pathologist, for examination. That report states that there was a middle section of the right lung which weighed 213 grams and measured 12.5 by 12.0 by 5.0 cm. EX 2:4.

In Dr. Caffrey's microscopic examination, he noted that slides "1" and "2" show some adhesions on the pleural surface with lobules of fat without inflammation or other specific atypia. On all ten slides there are only a few lesions of simple coal workers' pneumoconiosis (CWP), namely one lesion on slide "2", one lesion on slide "7", and three lesions on slide "6", for a total of five (5) lesions of simple CWP; the lesions of simple CWP therefore occupy less than five percent of the total lung tissue. On slide "4" there is a recent thromboembolus without infarction in a small vessel. On slide "8" there are two micro-nodules about 3 mm (0.3 cm) in size that show partial calcification and these appear to be silicotic nodules in Dr. Caffrey's opinion. Slide "9" and "10" show mucous and serous glands with approximately a three to one (3:1) ratio of mucous to serous glands with a mild infiltrate of mononuclear cells. There is small lymph node tissue on one of those slides that contain a few 1 mm (0.1 cm) silicotic nodules adjacent to the cartilage. There is no evidence of complicated pneumoconiosis. No asbestos and no ferruginous bodies are seen. EX 2:3. There is no evidence of pneumonia. Most of the slides show a moderate degree of centrilobular emphysema. EX 2:2-3.

Dr. Caffrey's Final Diagnosis was as follows: (1) Centrilobular emphysema, moderate; (2) Chronic bronchitis, mild to moderate; (3) Simple coal workers' pneumoconiosis, minimal; (4) Simple silicosis, minimal; and (5) Recent thromboembolus without infarction. EX 2:3. In elaborating on his diagnosis, Dr. Caffrey stated that Mr. Allen had a very mild or minimal degree of simple coal workers' pneumoconiosis and a minimal degree of simple silicosis. Dr. Caffrey opined that the amount of lung tissue involved by these diseases was less than five percent of Mr. Allen's lung issue, therefore this amount could not have caused him pulmonary disability. The amount of focal emphysema or centrilobular emphysema due to CWP was extremely minimal and would not disabling and the simple silicotic nodules did not cause emphysema. Dr. Caffrey noted that the surgical pathologist diagnosed macules and nodules compatible with simple CWP and emphysematous change. EX 2:4.

Dr. Caffrey also explained why he disagreed with Dr. Perper's opinion. Specifically, he disagreed that Mr. Allen had evidence of mild to moderately severe CWP with associated centrilobular emphysema. Rather, he believed that Mr. Allen had minimal or mild CWP.

[4] The ten slides were identified with the number "LP599-506" and on each slide the words "Highlands Pathology Kingsport, TN." EX 2:2. The slides are numbered "1-10" with the word "Recut" on each slide. EX 2:2. There is no reference to those numbers on the one-page report from Dr. Adelson (Dr. Caffrey received only ten slides, not eleven slides as indicated on Dr. Adelson's report). EX 2:2.

Moreover, Dr. Caffrey noted that he did not understand the phrase "mild to moderately severe" used by Dr. Perper. He asserted that if the CWP was mild it could not be moderately severe, and if it was moderately severe it could not be mild. EX 2:4. He also disagreed with any insinuation on the part of Dr. Perper that Mr. Allen's centrilobular emphysema was due to his simple CWP. Dr. Caffrey did not agree that individuals who have a significant degree of CWP can develop centrilobular emphysema. EX 2:4.Dr. Caffrey also undercut one of the sources cited by Dr. Perper. Specifically, he noted that Dr. Perper quotes from the "1998 second edition of … Diseases of the Chest by Frazer and Pare." However, Dr. Caffrey claimed that he owned the most recent edition of that textbook, copyrighted 1999, and that there was no 1998 second edition of which he was aware. He also noted that his 1999 copy was the fourth edition and that, while it listed the previous edition as 1988, it did not state whether that was the second or third edition. Dr. Caffrey also noted that, while his edition did include the statement quoted by Dr. Perper that "[i]n several investigations, emphysema has been shown to be present more often and to be more advanced in patients with CWP than in those without it," his edition did not include the next statement quoted by Dr. Perper that "the severity correlates with the degree of exposure to coal dust and has been demonstrated to be independent of age and cigarette smoking and to be positively related to the dust content of the macules." Dr. Caffrey reiterated that he did not agree that in individuals with a significant degree of simple CWP they can develop centrilobular emphysema. He restated that the slides showed only a minimal or mild degree and stated that "if there was any centrilobular emphysema present that is due to CWP it would be minimal or mild." EX 2:5.

Dr. Caffrey also criticized Dr. Perper for failing to allude to a study conducted by Drs. Lapp, Morgan, and Zaldivar entitled "Airways obstruction, coal mining, and disability," which was published in Occupational and Environmental Medicine, 1994. According to Dr. Caffrey, this study showed that out of a sample of 611 black lung claimants, there was only one subject who was a non-smoker and who, in the absence of other non-occupational related diseases, had "sufficient airway obstruction to render him difficult [sic] to carry out hard labor." EX 2:5.

In conclusion, Dr. Caffrey stated that Mr. Allen's medical problems were due to his years of smoking cigarettes for at least 50+ years. He stated that "every expert in this field knows that the number one cause of COPD is cigarette smoking" in asserting that Mr. Allen's disabilities, namely bronchitis and emphysema, were due to his years of smoking cigarettes. Dr. Caffrey also noted Mr. Allen's "significant vascular disease" in that he had had a myocardial infarction in 1996, which required coronary artery surgery, and after which Mr. Allen nevertheless developed congestive heart failure. Dr. Caffrey also noted that Mr. Allen had had diabetes mellitus, which accelerated his atherosclerotic disease. Finally, Dr. Caffrey noted that Mr. Allen's records demonstrated he had suffered "CVAs due to his atherosclerosis," which "certainly was not [sic] caused by or in any way influenced by his work in the coal mines." Dr. Caffrey stated that Mr. Allen's disabilities, pulmonary, cardiac, and cerebral, resulted from his years of smoking cigarettes; the fact that he had worked in the coal mines for 37 years did not cause, contribute to, or hasten his death, noting that Mr. Allen retired at age 62 and received a pension, as opposed to retiring because of a disability. EX 2:5.

Dr. Naeye's Opinion

Dr. Richard L. Naeye reviewed Mr. Allen's medical records on behalf of the Employer and provided a medical report dated January 18, 2003. EX 1. Dr. Naeye is a board-certified pathologist. EX 1:4. In connection with his report, Dr. Naeye provided an occupational history of over 37 years in underground mines working mostly as a belt operator or coal loader and a 37-40 year smoking history. He also reviewed interrogatory responses given by Mr. Allen, and Mr. Allen's medical records, including a copy of the biopsy report, glass slides with tissues removed postmortem, treatment records, a copy of the death certificate, and consultation reports from various occupational and pulmonary disease specialists, as well as Dr. Perper's report. EX 1:1. Dr. Naeye concluded that Mr. Allen's simple CWP was too mild to have caused significant impairments in lung function. Thus, it did not cause any of his disability or have any role in his death. Rather, his cigarette smoking, which began at age 12 and continued through most of his adult life, was responsible for the chronic bronchitis, bronchiolitis and progressive centrilobular emphysema that led to his disability and death. EX 1:3.

Dr. Naeye provided an in-depth explanation of his interpretation of the glass slides. He noted that he had received ten glass slides with lung tissues surgically removed postmortem from the middle lobe of Mr. Allen's right lung.[5] EX 1:1. Each of the ten slides had one large piece of lung tissue on it. EX 1:1. On two of these pieces of lung tissue were "three very old hyalinized nodules with a very tin rim of black pigment." EX 1:1-2. The largest of these old nodules was 8.5 mm long in one dimension. EX 1:2. Dr. Naeye reported: "There are no very [sic] tiny birefringent crystals of toxic free silica in any of the three lesions." EX 1:2. He further stated: "None have necrotic centers or an active proliferative process at their periphery that would identify them as lesions of complicated CWP." EX 1:2, c*iting* Kleinerman J, Green F, Laquer W, Taylor G, Harley R, Pratt P, Wyatt J, Naeye RL: Pathology standard for coal workers' pneumoconiosis, Arch Path & Lab Med, 103:375-432, 1979. Rather, Dr. Naeye noted "[t]hey appear to be very old healed granulomas of infectious origin." EX 1:2.

Dr. Naeye stated that "the ten pieces of lung tissue also have a small to moderate amount of black pigment in them in areas below the pleura and in small deposits adjacent to small arteries and airways." He further stated "there is sometimes a small amount of loose fibrous tissue admixed with the pigment as well as small numbers of moderate sized and large sized birefringent crystals of non-toxic silicates." He noted that "very tiny birefringent crystals of free silica are very rare." The black deposits just described are all one mm or less in diameter, which classifies them as anthracotic macules; some have a thin rim of surrounding focal emphysema. This latter emphysema constitutes less than 1% of the total emphysema in the lung tissues. Finally, Dr. Naeye reported that "[c]entrilobular emphysema varies from moderately severe to severe in the lung tissues; the ratio of mucus to serous glands in the walls of bronchi is about 10:1 (normal 1:1)." He concluded that "this is microscopic confirmation of very severe chronic bronchitis." EX 1:2.

[5] The slides were labeled LP99-506, recut, Highlands Pathology, Kingsport, TN. EX 1:1. Dr. Naeye also noted that an eleventh glass slide had lymph nodes. EX 1:1.

Next, Dr. Naeye critiqued Dr. Perper's medical report. Dr. Naeye asserted that, although Dr. Perper opined that Mr. Allen's progressive disability and eventual death resulted from his occupational exposure to coal mine dust, Dr. Perper's analysis actually "misrepresents the far more complex origin of [Mr. Allen's] progressive pulmonary disorders and eventual death." Specifically, Dr. Naeye noted that more than 85% of ex-coal miners have a long history of cigarette smoking, and observed that Dr. Perper neglected to mention the profound effects of this smoking when superimposed on exposure to mine dust. Dr. Naeye stated that Mr. Allen "without a doubt" had a progressive worsening of his chronic bronchitis and centrilobular emphysema as the years progressed both before and after he quit mining coal. EX 1:2. He opined that these two disorders, not CWP, were responsible for Mr. Allen's progressive disability and death, stating that the "anthracotic macules were far too small and few in number in his lungs to have caused any measurable abnormality in his deteriorating lung function." Dr. Naeye further stated, "[a]s will be seen, cigarette smoking usually has a much larger role in the genesis of bronchitis and emphysema than does bituminous mine dust." EX 1:2.

With regard to his specific criticisms of Dr. Perper's report, Dr. Naeye took issue with the sources that Dr. Perper cited to substantiate his belief that Mr. Allen's continued respiratory problems and eventual death resulted from the "natural course" of his CWP. *See* EX 1:2. Dr. Naeye observed that in setting forth this assertion, Dr. Perper cited British literature describing the advance of simple CWP to complicated CWP in that nation." However, according to Dr. Naeye, "[t]his is a very rare sequence in U.S. soft coal miners and it did not take place in Mr. Allen." Dr. Naeye further challenged Dr. Perper's citations to NIOSH literature. He contended that in "repeatedly attribute[ing] the progressive deterioration of lung function in many U.S. ex-bituminous coal miners to their exposure to mine dust," NIOSH has failed to note that most evidence demonstrating "the progression of CWP after leaving the mining industry is European in origin." According to Dr. Naeye, these "European citations are only rarely relevant to U.S. bituminous coal miners" and "[o]nly a rare U.S. bituminous coal miner has enough free silica in his lungs for CWP lesions to progress after he quits the industry. Dr. Naeye suggested that one reason for this may be that "more free silica is present in European than in U.S. bituminous mine dust." EX 1:2-3.

Moreover, Dr. Naeye pointed out that this literature often fails to "deal with the role of cigarette smoking in the long term deterioration of lung function in U.S. soft coal miners." According to Dr. Naeye, U.S. bituminous coal miners who never smoked cigarettes or who quit smoking very early in their lives almost never experience the long term deterioration in lung function experienced by Mr. Allen. Simple CWP advances mainly in the small number of U.S. bituminous miners who were exposed to large amounts of free silica during their working years. This latter group is most often comprised of roof bolters who drilled into free silica bearing rock above coal seams for many years. According to Dr. Naeye, such long exposures are rare in the U.S. EX 1:3.

Dr. Naeye noted that in Dr. Perper's 27-page report, he "devotes just 2.4 lines to his microscopic findings in the lungs of Mr. Allen." Dr. Naeye stated that, according to Dr. Perper, his microscopic review of the lung tissues "'verified the presence of simple, slight to moderately severe coal workers' pneumoconiosis, mostly macular and micronodular, with an occasional silicotic macronodule, scar emphysema and the presence of birefringent silica crystals.'" EX 1:2

citing DX 30. From this description, Dr. Naeye asserted, "it is perfectly clear that Dr. Perper has not made the diagnosis of complicated CWP in this case." Furthermore, Dr. Perper classified the CWP as "'slight to moderately severe.'" EX 1:2 *citing* DX 30. Dr. Naeye posited that Dr. Perper's designation of moderately severe "presumably comes from the two macronodules he thinks are silicotic in origin." By contrast, Dr. Naeye believed that his own "far more detailed examination and descriptions of these lesions" showed that most of the "silica crystals" Dr. Perper described as being present are non-toxic silicates. Specifically, the two macronodules Dr. Perper described "have no toxic free silica in them and thus clearly have a non-occupational origin." Dr. Naeye argued that if one subtracts these two "macronodules" from the CWP findings, "one is left with simple CWP that is far too mild to have caused any impairments in lung function or to have had any role in the death of this man." EX 1:2.

Dr. Naeye concluded by stating that Mr. Allen was a very heavy cigarette smoker for most of his life, and that it was his cigarette smoking, not mine dust exposure, that was mainly responsible for "the progressive disabling deterioration of lung function" that occurred in Mr. Allen. He restated that "U.S. bituminous miners who were non-smokers rarely experience progressive deterioration of lung function after they quit the industry," and that "studies of randomly selected populations of such miners have shown no effect of mine dust exposure on life expectancy." EX 1:3, c*iting* Foxman et al., Am Rev Resp Dis 1986, 134, :649, 1986; Ortmeyer C et al: Arch Environ Health 1974; 29:67. To that end, Dr. Naeye stated that "[s]uch expectancy would surely have been reduced if exposure to coal mine dust had caused clinically significant centrilobular emphysema, chronic bronchitis and bronchiolitis." In this latter regard, "coal mine dust exposure as well as smoking cigarettes can lead to chronic bronchitis, and less often to chronic bronchiolitis." However, multiple studies indicate that "in practice bronchitis has little or no effect on lung function unless the subject happens to be a smoker." EX 1:3, *Citing* Fletcher et al, The natural history of chronic bronchitis & emphysema, Oxford Press, 1976; Bates et al Am Rev Resp Dis 1973: 108:1043; Foxman et al Am Rev Resp Dis 1986.134:649. Airway obstruction caused by centrilobular emphysema and bronchitis that is severe enough to preclude a miner from working is very rare in the absence of smoking or complicated CWP. EX 1:3, *citing* Gautrin D. et al Exp Lung Research 20:395-410, 1994. Studies have also shown that chronic cor pulmonale does not occur in coal miners in the absence of cigarette smoking, *citing* Fernie, Douglas et al, Thorax 1983, 38:436. Finally, he said, there is strong evidence that simple CWP does not progress after a miner leaves the industry, *citing* Attfield MD, Althouse R, Hall B, Kellie S: Coal study and related research. Final report from the round 3 of the National Coal Workers Pneumoconiosis Study, PB 85-221026, 1984; Althouse, R, Attfield MD, Kellie S. Use of data from x-ray screening program for coal workers to evaluate effectiveness of the 2 mg/m3 coal dust standard. J Occupat Med 28:741-745, 1986.; Gautrin D et al: Exp Lung Res 1994:20:395; Attfield & Hodus, Am Rev Resp Dis 1992, 145:605.

Dr. Thomashefski's Review of the Post Mortem Biopsy Slides

Dr. Joseph T. Thomashefski reviewed the autopsy slides on behalf of the Employer and provided a report dated September 8, 2003. EX 3. Dr. Thomashefski is a board-certified pathologist in anatomic and clinical pathology. In preparing his report, he reviewed Mr. Allen's medical history, including treatment records and medical reports of other physicians. Dr.

Thomashefski also reviewed ten slides of lung tissue labeled LP99-506, which were of "satisfactory technical quality." EX 3:1.

Dr. Thomashefski stated that, based on his review of the medical records and slides of the lung tissue, he believed that Mr. Allen had had "mild centriacinar and paraseptal emphysema associated with pleural adhesions, mild non-specific pleural fibrosis, mainly associated with paraseptal emphysema, and mild focal organizing pneumonia." He further opined, based on the finding of scattered coal macules associated with focal emphysema, that Mr. Allen had had mild simple coal workers' pneumoconiosis. In addition, Mr. Allen had had at least one silicotic nodule in his peripheral lung and several nodules in the peribronchial hilar lymph nodes. EX 3:5.

As the autopsy performed on Mr. Allen was limited to a postmortem biopsy of his right lung, Dr. Thomashefski could not determine, within reasonable medical certainty, the anatomical cause of his death. EX 3:5. He noted that the medical records suggest that his death was due to a combination of pneumonia and congestive heart failure and that his lung tissue demonstrated focal organizing pneumonia as well as bronchial lymphangiectasia, consistent with his history of pneumonia and left ventricular cardiac failure. EX 3:5. Dr. Thomashefski opined, however, that the degree of simple coal workers' pneumoconiosis in Mr. Allen's lung tissue was too mild to have caused him any respiratory symptoms or significant respiratory impairment; in other words, the mild degree of simple CWP neither caused nor contributed to Mr. Allen's death. EX 3:6.

Dr. Thomashefski observed that Dr. Perper had opined that Mr. Allen had had evidence of mild to moderately severe CWP with associated centriacinar emphysema, and that CWP with related centriacinar emphysema substantially contributed to Mr. Allen's death. EX 3:6. While Dr. Thomashefski agreed with Dr. Perper's diagnosis of simple CWP and centrilobular emphysema, he reiterated that the extent of each of these conditions was mild. EX 3:6. Dr. Thomashefski further asserted that his opinion was supported by Mr. Allen's pulmonary function test results, which were either in the normal range, or at most showed only mild obstructive changes. The last arterial blood gas value (8/7/86) prior to Mr. Allen's prolonged terminal illness was also normal. He also pointed out that his opinion regarding Mr. Allen's mild simple CWP was supported by chest radiographic interpretations of experienced B-readers, many of whom found no radiographic evidence of CWP. EX 3:6.

In a similar vein, Dr. Thomashefski disagreed with Dr. Perper's conclusion that Mr. Allen's mild centriacinar emphysema was due to coal dust exposure. He noted that the lesions of centriacinar emphysema, for the most part, bore no spatial association with the few coal macules that were present. In his opinion, Mr. Allen's mild centriacinar emphysema was caused by heavy and sustained exposure to cigarette smoke. In that regard, Dr. Thomashefski believed that the degree of CWP and centriacinar emphysema in Mr. Allen's lung tissue was too mild to have been a cause or a contributory factor in his death. He stated that "within reasonable medical certainty, Mr. Allen's death was secondary to pneumonia and/or cardiac failure, neither of which was caused by coal dust exposure or mild simple CWP." Finally, Dr. Thomashefski stated that Mr. Allen would have died at the same time and in the same manner even if he had never been a coal miner or developed mild simple CWP. EX 3:6.

Dr. Castle's Opinion

Dr. James R. Castle also reviewed Mr. Allen's records on behalf of the Employer, and issued a medical report dated September 22, 2003. EX 5. Dr. Castle is a board-certified in internal medicine and in the subspecialty of pulmonary diseases, and a B reader. He has practiced pulmonary medicine in Roanoke, Virginia, since 1977. In preparing his report, Dr. Castle reported an occupational history of 37 years, ending in 1980. After reviewing the "significantly variable" smoking histories in the record, he assessed a 50 pack-year smoking history. Dr. Castle also reviewed Mr. Allen's medical history, including treatment records and medical reports of other physicians. *See* EX 5:1-22.

Dr. Castle opined that Mr. Allen did have pathologic evidence of simple CWP based upon a thorough review of all the data, including the medical histories, physical examinations, radiographic evaluations, physiologic testing, arterial blood gases, hospital records, autopsy material, and other data. Dr. Castle noted that Mr. Allen's work in the underground mining industry was sufficient to have caused him to develop CWP if he were a susceptible individual. However, Dr. Castle also noted that tobacco abuse is another risk factor for the development of pulmonary symptoms and disease. According to Dr. Castle, Mr. Allen's smoking history was sufficient to have caused him to develop chronic obstructive pulmonary disease, *i.e.* chronic bronchitis/emphysema and/or lung cancer and/or atherosclerotic cardiovascular disease if he were a susceptible host. Moreover, Dr. Castle noted that another risk factor for the development of pulmonary symptoms is that of coronary artery disease. In that regard, Mr. Allen had had a documented history of coronary artery disease and had required coronary artery bypass grafting. EX 5:22. In fact, it was noted that he had had an ischemic cardiomyopathy and recurrent episodes of congestive heart failure. Mr. Allen was also "troubled intermittently by tachycardia-bradycardia arrhythmia syndrome occurring periodically." EX 5:22-23. Dr. Castle asserted that these types of cardiac disease, coronary artery disease, ischemic cardiomyopathy with congestive heart failure, and tachycardia-bradycardia arrhythmia syndrome, are unrelated to coal mine dust exposure and CWP. Dr. Castle stated that valid physiologic studies showed, at worst, minimal airway obstruction without restriction or diffusion abnormality. Mr. Allen did not demonstrate valid objective findings indicating pulmonary impairment prior to the development of significant congestive heart failure and ischemic cardiomyopathy. EX 5:23.

Dr. Castle stated that at no time did Mr. Allen demonstrate any physical findings indicating the presence of an interstitial pulmonary process; he did not demonstrate a consistent finding of rales, crackles, or crepitations. He further stated that "there was a significant disparity in the radiographic interpretations for the presence or absence of CWP," and that in his opinion Mr. Allen "probably did have radiographic evidence consistent with minimal, simple CWP." Dr. Castle noted that the chest x-rays obtained on July 17, 1999, and later, did not indicate worsening of changes related to any underlying pneumoconiosis. At that time, Mr. Allen had developed Methicillin resistant Staphylococcus pneumonia with resultant ARDS (Adult Respiratory Distress Syndrome), which is a disease of the general public at large and is totally unrelated to coal mining employment and coal dust exposure. Dr. Castle stated that these problems persisted over the ensuing several months, and ultimately culminated in chronic respiratory failure associated with congestive heart failure. EX 5:23.

While he concluded it "very likely" that Mr. Allen's chest x-ray did demonstrate evidence of minimal, simple CWP (observing that all of the pathologists who reviewed the autopsy tissues felt there to be evidence of simple CWP), Dr. Castle also noted that complicated CWP was not described. EX 5:23-24. In that regard, Dr. Castle noted that, while Dr. Perper described mild-moderately severe changes, Dr. Naeye described in great detail why there was only evidence of mild CWP pathologically. EX 5:24. If abnormal arterial blood gas studies had been due to CWP, they would not have improved at a later date. He agreed with others that the earlier hypoxemia was due to Mr. Allen's obesity at the time. EX 5:24. Dr. Castle opined that Mr. Allen did not demonstrate a disabling abnormality of blood gas transfer mechanisms which was related to occupational pneumoconiosis or his coal mine dust exposure. EX 5:24. He found it "absolutely clear" that Mr. Allen developed significant respiratory failure with hypoxemia after July 1999 due to the severe pneumonia and ARDS which resulted from the severe pneumonia. Mr. Allen was unable to be weaned from a ventilator because of multiple medical problems including ARDS, severe pneumonia, probable tobacco induced chronic obstructive pulmonary disease, and congestive heart failure. EX 5:24.

Dr. Castle opined that Mr. Allen's death was neither caused by, contributed to, nor hastened by the underlying CWP that was present pathologically. Rather, Dr. Castle believed that Mr. Allen's death was caused by complications from Methicillin resistant Staphylococcus aureus pneumonia including ARDS with respiratory failure. Dr. Castle stated that the nurses' notes and other records indicate that Mr. Allen "expired very suddenly most likely due to the cardiac arrhythmia." Moreover, Dr. Castle opined that the cardiac arrhythmia was due to Mr. Allen's underlying severe atherosclerotic cardiovascular disease with ischemic cardiomyopathy. Mr. Allen had had previously documented severe cardiac arrhythmias including ventricular tachycardia; these were unrelated to CWP or coal mine dust-induced lung disease. Dr. Castle asserted that these are all conditions of the general public at large and are unrelated to coal mining employment, coal dust exposure, and CWP. Dr. Castle also opined that Mr. Allen did not have a disabling respiratory impairment during life due to CWP. In 1986, approximately six years after leaving the mining industry, Mr. Allen's ventilatory function was still essentially within normal limits. Thus, Dr. Castle opined that any respiratory impairment which developed subsequent to that would have been unrelated to CWP. Further, Dr. Castle stated that Mr. Allen did have evidence of "tobacco smoke induced chronic bronchitis and pulmonary emphysema as was diagnosed pathologically." He noted that as late as 1986, Mr. Allen did not demonstrate a disabling respiratory impairment. EX 5:24. Finally, Dr. Castle opined that Mr. Allen would have died as and when he did regardless of his occupational history and regardless of the presence of CWP. EX 5:24-25.

In addition to providing his September 22, 2003 medical report, Dr. Castle also testified by way of deposition on October 6, 2003. EX 7. As he had done in his September 22, 2003 medical report, Dr. Castle recited Mr. Allen's occupational history and smoking history. Also, in preparation for his deposition, Dr. Castle reviewed additional medical reports concerning Mr. Allen, including the pathology report by Dr. Thomashefski dated September 8, 2003. EX 7:9-10.

Dr. Castle testified that, in sum, Mr. Allen's blood gases and pulmonary function studies had been normal in 1986, at which time Mr. Allen was in his late sixties. EX 7:13. Based on his

review of the record, Dr. Castle noted that Mr. Allen had had a number of pulmonary function studies performed up until about 1986. EX 7:11. At that time, the very worst that he had experienced was "very minimal airway obstruction without restriction or diffusion abnormality." EX 7:11-12. In August 1986, Dr. Dahhan examined him and performed pulmonary function studies, as well as resting and exercise blood gases. Dr. Dahhan found him to have normal ventilatory function without any evidence of obstruction or restriction, and in 1986, six years after leaving the mining industry, even the MVV was normal. Thus, Mr. Allen did not have any respiratory impairment noted in 1986, despite his history of heavy smoking. EX 7:12.

Dr. Castle further testified that Mr. Allen's blood gases in the 1980s were generally conducted in association with a disability evaluation and were found to be "variable." He stated: "I think that Dr. Robinette in 1985 found a qualifying study with a pO2 of 60.5 mm of mercury at rest with a pO2 in the '30s. EX 7:12. Subsequent to that, in 1986, Dr. Dahhan found a normal resting arterial blood gas study with a normal response to exercise. EX 7:12-13. Dr. Castle stated:

> Now, that clearly shows that he had some variability in his oxygenation and that it was not – that this degree of hypoxemia noted by Dr. Robinette on a given day was not a fixed abnormality. It means that in 1986, he had normal function and normal response to exercise, and the change noted by Dr. Robinette could not have been due to any coal workers' pneumoconiosis, and the reason is quite simple. Coal workers' pneumoconiosis is an irreversible process, and when it causes impairment, it is not variable and does not change with time and treatment.

EX 7:13.

Dr. Castle noted that this was the most recent physiologic testing and arterial blood gas testing that gad taken place until 1999, when Mr. Allen developed a very severe illness. Dr. Castle testified that all of the blood gases conducted in July, August, and September of 1999 were performed in connection with an acute problem/hospitalization. EX 7:13. Specifically, Mr. Allen was initially hospitalized on July 11, 1999, when he was found to have pneumonia. EX 7:13-14. He very rapidly developed a condition known as adult respiratory distress syndrome (ARDS), which is a "severe devastating form of respiratory failure." Dr. Castle testified: "Those blood gases were subsequent to his developing that degree of condition associated with pneumonia and indicated just that he had those conditions, severe respiratory failure due to a severe acute pneumonia." EX 7:14.

Dr. Castle testified that he reviewed the notes of Dr. Gary Williams, Mr. Allen's primary care physician, which pertained to 1996 through 1999. Dr. Williams did not perform blood gases, but Mr. Allen's pulse oximetry, which is a reflection of his oxygenation, was 93 percent at rest and 91 percent with exercise. Dr. Williams gave him a prescription for oxygen, but noted that he did not think Mr. Allen's insurance would cover it, which Dr. Castle believed would have been the case since "those saturations are essentially normal." EX 7:14. However, Dr. Williams noted that he felt it would "be very reassuring to Mr. Allen and would help to lower his level of anxiety." EX 7:14-15. In sum, Dr. Castle stated that Mr. Allen had had normal oxygen saturation in June 1999, prior to the development of pneumonia in July 1999. EX 7:15.

With regard to what happened to Mr. Allen during the last four to six months of his life, Dr. Castle noted, preliminarily, that in 1998, Mr. Allen had already developed problems with cardiomyopathy and ischemic heart disease (*i.e.* coronary heart disease), and had experienced episodes of congestive heart failure and required hospitalization. EX 7:15 Later, in July 1999, he presented to the hospital in Norton with right lower lobe pneumonia, and then very rapidly developed these diffuse pulmonary infiltrates, which were initially thought to be possibly due to heart failure. EX 7:15. However, Dr. Castle testified that over time, it became apparent that they were due to adult respiratory distress syndrome ("ARDS"). EX 7:15-16. As a result, Mr. Allen was hospitalized at Norton but transferred to Bristol Regional Medical Center for treatment of his severe respiratory failure and ARDS. Ultimately, Mr. Allen developed progressive problems related to renal problems and diabetes, and subsequently had experienced multiple strokes with multi-infarct dementia. He required a tracheostomy and "really was not adequately weaned from a ventilator, was ventilator dependent, and had resistant Staph aureus pneumonia." EX 7:16.

Dr. Castle testified that Mr. Allen continued to have these extensive infiltrates, which were not due to CWP or emphysema, but were rather due to ARDS caused by the pneumonia. Ultimately, Mr. Allen was sent to a nursing home. After being sent to the nursing home, Mr. Allen experienced an episode of tachycardia-bradycardia arrhythmia, and his heart rate went down to the 30's. He was sent to the hospital, and there they found that he was having episodes of ventricular tachycardia. Thus, Dr. Castle testified, Mr. Allen was having cardiac arrhythmias related to his coronary artery disease and his ischemic cardiomyopathy. Mr. Allen was subsequently sent back to the nursing home and stabilized. EX 7:16. On the date of his death, September 26, 1999, the nurses' notes indicate that he had "absent vital signs" at 12:45 PM. Earlier in the day, he had been awake and alert and nodded his head appropriately to yes and no questions. Mr. Allen was found "essentially dead" at 12:45 PM, and was taken to the hospital to be pronounced. The family requested an autopsy of the lungs. Dr. Castle testified that Mr. Allen died suddenly, most likely as a result of his cardiac arrhythmia. In that regard, Dr. Castle noted that Mr. Allen had been experiencing ventricular tachycardia, which had been noted in the hospital, and thereafter was found to have died suddenly. EX 7:17.

When questioned as to whether cigarette smokers have a higher incidence of cardiac arrhythmia than non-smokers, Dr. Castle testified: "It's not related to cigarette smoking per se; it's related to atherosclerotic cardiovascular disease, and certainly, people that are cigarette smokers have a much higher incidence of that situation, and it is a risk factor for coronary artery disease, so that answer to that would be yes." EX 7:17.

Dr. Castle testified that Mr. Allen's autopsy was limited to the lungs. EX 7:17-18. He further stated that a biopsy of the right lung was performed. Dr. Adelson was the pathologist who would have been the prosector, and he diagnosed simple CWP with some emphysematous changes being present as well. Drs. Perper, Naeye, and Thomashefski confirmed a finding of simple CWP after reviewing the slides. EX 7:18.

When questioned as to how extensive the pneumoconiosis was, Dr. Castle testified that Dr. Adelson "did not indicate that it was severe," and Drs. Naeye and Thomashefski both indicated that it was "very mild and did not involve large amounts of lung tissue." Dr. Castle

also noted Dr. Perper's opinion that there was "mild to moderately severe coal workers' pneumoconiosis." Accordingly, Dr. Castle testified that, based upon the descriptions of all of the pathologists, he would have to conclude that it was "very mild." EX 7:18. Dr. Castle observed that this would be confirmed by the fact that Mr. Allen did not have any significant respiratory impairment related to pneumoconiosis, as demonstrated by the normal physiological function that he demonstrated during life. EX 7:19.

Dr. Castle testified that CWP played "no role at all" and it "certainly did not hasten [Mr. Allen's] death." He further stated that Mr. Allen had "very severe cardiac disease with ischemic cardiomyopathy. He then developed a pneumonia which resulted in a complication called adult respiratory distress syndrome. Dr. Castle stated that adult respiratory distress syndrome is a disease "totally unrelated to coal dust and CWP." He opined that if Mr. Allen had been "anything else" (*i.e.* not a coal mine worker), "this would have happened the same way." He further stated: "[ARDS] is a condition we seen in anybody, and anybody can develop ARDS." Dr. Castle explained that there is no way to predict who will and who will not get ARDS. In other words, it is not related to any underlying disease process. EX 7:19. Dr. Castle explained that, today, ARDS is commonly related to sepsis or pneumonia or any kind of blood-borne infection, and it is an inflammatory process. It normally has a mortality rate of approximately 20 to 30 percent, depending on the expertise involved. EX 7:20.

With regard to Mr. Allen's case, Dr. Castle opined that the "doctors in Bristol are very good" and that he believed "what we are dealing with here is a man that had the severe part of his syndrome and died as a result of that." Moreover, he testified that the fact that Mr. Allen was 81 years old and had had severe cardiac disease "did not help." EX 7:20. He stated "[t]hat means you've got now two organ systems that are failing. You have the heart that is failing and the lungs that are failing because of this acute inflammatory process. The heart, of course, is failing because of coronary artery disease, and now, when you take those two major organs that are failing, it certainly increases the mortality generally to around 60 to 70 percent." EX 7:20-21.

On cross-examination, Dr. Castle admitted that Mr. Allen's death was caused at least in part by respiratory problems. At the same time, he reiterated his position regarding the role of pneumonia and ARDS. He explained that pneumonia is an infection. In this case, Mr. Allen's pneumonia was due to bacteria known as methicillin resistant staphylococcus aureus ("staph"); essentially, Mr. Allen had "pus cells" in his lung. Dr. Castle testified that the staph germ is resistant to penicillin and like drugs, and therefore can be treated only by one or two specific drugs. This "inflammatory process" then causes the release of chemical mediators in the bloodstream, which results in ARDS, which results in respiratory failure. Specifically, "the chemical mediators cause the destruction of the lung tissue by allowing material to seep out through holes that occur in the alveolar walls." The material that "seeps out" has "a lot of protein" and "inflammatory cells in it," which "in and of itself" causes respiratory failure as the alveoli become "filled up" with this "thick fluid with protein in it," causing the lungs to become "very still." As a result, "the patient cannot breathe very well, and they generally require mechanically assisted ventilation … to try to improve their oxygenation." Dr. Castle testified that nothing specific had ever been developed to treat this process; rather, the typical approach is to "carry the patient along until they can get over it." He explained that in some cases it will

resolve, while in others it causes "persistent chronic infiltrates," and ultimately may develop "hyalin membranes" in the lung and fibrosis with the resultant scar tissue." In such a case, the patient never gets off the ventilator and dies. EX 7:22-24.

On cross examination, Dr. Castle reiterated that, based upon the pathology reviews, he felt that Mr. Allen had had mild simple CWP, "further confirmed by the fact that he did not have any respiratory impairment due to that." EX 7:24-25. With regard to how much of the lungs are affected by mild simple CWP, Dr. Castle testified that Dr. Thomashefski had indicated no more than one or two percent. He further stated: "[T]hese were widely spaced macules that were not associated with most of the centriacinar emphysema that was present, and in the range of one or two percent of the lung tissue would have been affected by that." EX 7:25. When questioned on cross-examination as to whether this tissue would no longer function as lung tissue in the sense that it would no longer operate to exchange oxygen, Dr. Castle pointed out that the lung tissue around it would "certainly be able to function" normally, since it had been doing so until July 11th (1999) when Mr. Allen developed pneumonia and ARDS. Dr. Castle stated that prior to that time, "it had been functioning very well," noting that in late June 1999, Mr. Allen's oximetry was entirely normal, and his doctor had indicated that he "didn't really need oxygen." EX 7:25-26. Nevertheless, Dr. Castle admitted that the one to two percent of the lung tissue that had been affected by the simple CWP would not function as lung tissue. With regard to whether the pus essentially "drowned" Mr. Allen, Dr. Castle testified: "Well, I think that you could say the ARDS caused him to be unable to oxygenate well, and that is what happens in drowning." EX 7:26.

Dr. Castle testified that when Mr. Allen presented to the hospital on July 11th (1999), he had had a right lower lobe infiltrate and pneumonia. This meant that the pneumonia itself was confined to the right lower lobe, which is where he would have had these pus cells. The inflammatory response, which is a systemic event throughout the entire body, involves the entire aspect of both lungs. Thus, a day later, Mr. Allen had "diffuse infiltrates in both lungs as a result of ARDS." According to Dr. Castle, this is "what got him into severe respiratory failure very quickly, very suddenly." Dr. Castle further opined that if Mr. Allen had not developed ARDS, he would have had "a totally treatable pneumonia," which would have been difficult to treat, but was certainly limited to an area of the one lung." EX 7:27.

Thereafter, Dr. Castle was questioned as to whether, since one to two percent of Mr. Allen's lung tissue was not functional due to CWP, and since he was "drowning in pus," then would he have "drowned" one percent faster because he had one percent less lung tissue to drown due to his CWP. EX 7:27-28. Dr. Castle explained that this would not have been the case because "of the reserve that is present in the lung." He further stated: "That one percent played no role at all. He would have been the same whether he had that one percent or not. It would not have made any difference in any way at all." EX 7:28. Dr. Castle reiterated: "[T]hat one percent would not modify or change the fact that this man had severe respiratory failure as a result of some other process. Even if he had … one percent of T.B., that would not have played any role either, simply because the disease process that he had was a terminal event in conjunction with his heart problem." EX 7:29. Finally, Dr. Castle stated: "[H]e died of a cardiac arrhythmia and died suddenly. There is no way that this one percent could have caused him to die any quicker or live any longer … it didn't play any role at all." EX 7:29-30.

Discussion

The regulations define pneumoconiosis broadly:

> (a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis.
>
> (1) Clinical Pneumoconiosis. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silico-tuberculosis, arising out of coal mine employment.
>
> (2) Legal Pneumoconiosis. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.
>
> (b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.
>
> (c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 CFR § 718.201 (2005).

In this case, the post mortem lung biopsy conclusively establishes that Mr. Allen had simple clinical pneumoconiosis. While all of the physicians who have given opinions agree on this point, none have diagnosed complicated pneumoconiosis. Thus the irrebuttable presumption that death was due to pneumoconiosis set forth in 20 CFR § 718.304 does not apply. Moreover, although the death certificate identifies pneumoconiosis as a contributing cause of Mr. Allen's death, it is not supported by a reasoned explanation from any of his treating physicians. Thus the case must be decided by weighing the conflicting medical opinions of Dr. Perper, a pathologist, that pneumoconiosis did contribute to his death, on the one hand; and the contrary views of Drs. Caffrey, Naeye and Thomashefsi, all pathologists, and Dr. Castle, a pulmonologist, on the other.

Looking first to the pathologists, the sheer weight of numbers taking the position that pneumoconiosis did not hasten Mr. Allen's death suggests that weight of the pathological

evidence is against the Claimant's position. All of the pathologists who provided opinions are well-qualified to do so, and had extensive documentation available to them. The opinions offered by the pathologists consulted by the Employer, however, suffer from similar flaws: they focus on the diagnosis and effects of clinical pneumoconiosis, to the exclusion of the broader definition of legal pneumoconiosis; and, they give no credence to the premise underlying the Department of Labor's regulations, *see* the commentary that accompanied the final version of the current rules found at 65 Fed. Reg. 79920, 79938-79943 (2000), that coal dust exposure may induce obstructive lung disease and concomitant lung impairment even in the absence of simple or complicated clinical pneumoconiosis. The Department of Labor has concluded that "[e]ven in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis. The risk is additive with cigarette smoking." 65 Fed. Reg. at 79940. None of the pathologists retained by the Employer acknowledged the combined effects of smoking and coal mine dust exposure.

Dr. Caffrey disagreed with what he called Dr. Perper's inference that Mr. Allen's centrilobular emphysema was due to simple CWP. Although Dr. Caffrey agreed that "individuals who have a *significant* degree of CWP can develop centrilobular emphysema," he went on to state that because the slides showed only a minimal or mild degree of pneumoconiosis, "if there was any centrilobular emphysema present that is due to CWP it would be minimal or mild." EX 2 at pages 4-5 of the report (emphasis added). He attributed virtually all of Mr. Allen's medical problems to smoking. Dr. Caffrey did not offer a specific opinion on the cause of death; rather, he stated only the negative, i.e., that Mr. Allen's work in coal mines "did not cause, contribute to, or hasten his death." EX 2, page 5 of the report.

Similarly, Dr. Naeye took the position that absent a diagnosis of complicated pneumoconiosis, Mr. Allen's simple CWP was "far too mild to have caused any impairments in lung function or to have had any role in the death of this man." EX 1, report at page 2. He criticized Dr. Perper for failing to mention the role of smoking. He said that Mr. Allen's cigarette smoking "was responsible for the chronic bronchitis, bronchiolitis and progressive centrilobular emphysema that led to his disability and death." Ibid. at page 3. Dr. Naeye also challenged whether mine dust progresses after a miner leaves the mines, contrary to the Department of Labor findings and another tenet underlying the current regulations. *See* 65 Fed. Reg. at 79970-79971 (2000).

Dr. Tomashefski discounted any relationship between Mr. Allen's emphysema and coal dust exposure because, "for the most part, [the lesions] bear no spatial association with the few coal macules that are present." EX 3, report at page 6. He, too, attributed all of Mr. Allen's emphysema to smoking. On the other hand, he also said that because the autopsy was limited to the lung biopsy, he could not "determine, within reasonable medical certainty, the anatomical cause of his death. The medical records suggest that his death was due to a combination of pneumonia and congestive heart failure." Ibid. at page 5. He went on to state that in his opinion, "Mr. Allen would have died at the same time and in the same manner even if he had never been a coal miner or developed mild simple coalworkers' pneumoconiosis." Ibid. at page 6.

Because their opinions are based on general premises contrary to the Department's findings underlying the rules, all three lose probative value. On the other hand, while Dr. Perper,

like the other pathologists, focused on the evidence of clinical pneumoconiosis from the post mortem lung biopsy, his report makes clear that he accepts that exposure to coal mine dust, as well as smoking, can contribute to emphysema and chronic obstructive pulmonary disease, with or without evidence of clinical pneumoconiosis. For this reason, I give greater weight to the opinion of Dr. Perper among the pathologists.

In formulating his opinion, Dr. Perper acknowledged that Mr. Allen had a 40-pack-year smoking history. I do not find that there is any material difference between the 40-pack-year history acknowledged by Dr. Perper, and the 50-pack-year history found by other doctors. All of the doctors, including Dr. Perper, recognized that Mr. Allen quit smoking after he left the mines. Thus I do not credit the criticism that Dr. Perper failed to take Mr. Allen's smoking history into account. However, the probative value of Dr. Perper's report on the cause of death is reduced because it focused most extensively on the issue of whether Mr. Allen had pneumoconiosis, which the Employer had conceded by the time the case was referred for hearing. Dr. Perper also stated that pneumoconiosis contributed to Mr. Allen's death through its role in causing hypoxemia resulting in respiratory failure, and through its role in precipitation of a fatal heart arrhythmia, but gave no more explanation of the reasons for his opinion; nor did he explain it specifically in the context of Mr. Allen's last illness. Although Dr. Perper's discussion of the cause of death was less detailed than his discussion of the diagnosis, it would nonetheless be sufficient to support a finding that pneumoconiosis hastened Mr. Allen's death, absent a more compelling explanation to the contrary. The inquiry does not end here, however, as I must also consider the opinion of Dr. Castle, the pulmonologist.

Dr. Castle has extensive qualifications based upon his credentials, and upon his medical practice, teaching and research. *See* his deposition, EX 7, at pages 4-8, and Curriculum Vitae, Deposition Exhibit 2. Dr. Castle agreed that Mr. Allen had simple (clinical) coal workers' pneumoconiosis. Based on physiological studies, Dr. Castle concluded that Mr. Allen had "at worst minimal airway obstruction without restriction or diffusion abnormality" and occasional mild hypoxemia which improved on later testing. EX 5, report at page 23. Based on this data, observing that Mr. Allen "had essentially normal physiologic function after leaving the mining industry," ibid., Dr. Castle concluded that Mr. Allen did not have a pulmonary or respiratory impairment due to his work in coal mining, or from any cause, as late as 1986. He said Mr. Allen

> did develop significant respiratory failure with hypoxemia after July 1999 due to the severe pneumonia and ARDS which resulted from the severe pneumonia. He also was unable to be weaned from a ventilator because of multiple medical problems including ARDS, severe pneumonia, probable tobacco smoke induced chronic obstructive pulmonary disease, and congestive heart failure.

Report at page 24. As to cause of death, Dr. Castle stated in his report,

> It is my opinion with a reasonable degree of medical certainty based on a through review of all the data that Mr. Charles Allen's death was neither caused by, contributed to, nor hastened by the underlying coal workers' pneumoconiosis *that was present pathologically*. It is my opinion with a reasonable degree of medical certainty that his

> death was caused by complications from Methicillin resistant Staphylococcus aureus pneumonia including Adult Respiratory Distress Syndrome with respiratory failure. The nurses notes and other records would indicate that he expired very suddenly most likely due to the cardiac arrhythmia. It is my opinion that this cardiac arrhythmia was due to his underlying severe atherosclerotic cardiovascular disease with ischemic cardiomyopathy. He had documented previous severe cardiac arrhythmias including ventricular thachycardia. These were unrelated to coal workers' pneumoconiosis or coal mine dust induced lung disease. … It is my opinion with a reasonable degree of medical certainty that Mr. Allen would have died as and when he did regardless of his occupational history and regardless of the presence of coal workers' pneumoconiosis."

Report at pages 24-25 (emphasis added). The emphasized language from the report suggests that Dr. Castle, like the pathologists, focused on the role of clinical, but not legal, pneumoconiosis, in Mr. Allen's condition. During his deposition, however, in response to a question whether coal workers' pneumoconiosis played any role in Mr. Allen's death, Dr. Castle responded as follows:

> No, sir, it played no role at all, and it certainly did not hasten his death. This gentleman had very severe cardiac disease with ischemic cardiomyopathy and then unfortunately developed a pneumonia which resulted in a complication called adult respiratory distress syndrome. That is a disease totally unrelated to coal dust and coal workers' pneumoconiosis, and if this man had been anything else, this would have happened the same way. This is [a] condition that we see in anybody, and anybody can develop ARDS.
>
> There is no way to predict who will or will not get it, *so it's not related to any underlying disease process.* …
>
> …
>
> Today, it is … commonly related to sepsis or pneumonia or any kind of blood-borne infection, and it is an inflammatory process and it has a mortality of approximately 20 to 30 percent … and I think that what we are dealing with here is a man that had the severe part of this syndrome and died as a result of that. …
>
> …
>
> The fact that he was 81 years old and had severe cardiac disease did not help. That means that you've got now two organ systems that are failing. You have the heart that is failing and the lungs that are failing because of this acute inflammatory process. … [I]t certainly increases the mortality generally to around 60 or 70 percent.

EX 7, Deposition at 19-21 (emphasis added). In response to additional questions, Dr. Castle said that a small reduction in lung capacity due to underlying disease made no difference to the course of his final illness. EX 7, Deposition at 27-30. These responses appear to rule out any role for chronic obstructive disease in Mr. Allen's death, regardless of what caused it. The Claimant did not offer any evidence to rebut Dr. Castle's report and testimony.

Weighing Dr. Perper's opinion against Dr. Castle's, I conclude that the Claimant has failed to bear her burden to establish that pneumoconiosis contributed to Mr. Allen's death. Dr. Castle gave a much more thorough and complete explanation of the course of Mr. Allen's final illness and the role of any lung impairment in his death. His opinion is consistent with Mr. Allen's treatment records and the objective evidence in the record. Dr. Castle's opinion provides convincing evidence that although Mr. Allen had pneumoconiosis, it did not hasten his death. For this reason, I give it more weight than Dr. Perper's. As the Claimant had the burden of proof on this issue, her claim must fail.

FINDINGS AND CONCLUSIONS REGARDING ENTITLEMENT TO BENEFITS

Because the Claimant has failed to meet her burden to establish that pneumoconiosis hastened Mr. Allen's death, she is not entitled to benefits under the Act.

ATTORNEY FEES

The award of an attorney's fee under the Act is permitted only in cases in which the claimant is found to be entitled to benefits. Section 28 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 928, as incorporated into the Black Lung Benefits Act, 30 U.S.C. § 932. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for services rendered to her in pursuit of this claim.

ORDER

The claim for benefits filed by Geneva Allen on February 1, 2001, is DENIED.

A

ALICE M. CRAFT
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).